plications which are usually the direct cause of death of patients suffering from the malady.

It has been observed and confirmed that in certain instances of this disease human tubercle bacilli may be present in lymph glands which are not the seat of tubercle formation. This may be explained by the unique "hyper-allergic" reaction of these patients to tuberculin. In such instances we should expect marked constitutional reaction with little tubercle formation.

SAMUEL AYERS, JR., M. D. (Westlake Professional Building, Los Angeles)—The acute disseminated type of lupus erythematosus is a rare disease and my personal experience with it is meager. One of the most striking facts is the apparent inadequacy of pathological findings. Two recent cases with necropsy findings reported by Keefer and Felty showed abdominal tuberculous glands as the only pathological findings of note, and in one of these cases tubercle bacilli of the human type were recovered from a lymph gland, which did not show the histologic lesions characteristic of tuberculosis.

Foci of infection of a non-tuberculous type certainly play an important etiological role in the ordinary subacute or chronic types of lupus erythematosus. I have one striking example of this in a woman who developed a rather acute facial lupus erythematosus with classical symptoms, in whom chronically infected tonsils were found After persisting some months the eruption cleared immediately following tonsillectomy—in fact before the patient had fully recovered from the operation—and has not recurred during the past three years. All possible foci of infection should be searched for in all types of lupus erythematosus—tonsils, teeth, sinuses, prostate, intestinal tract, kidneys, bladder, etc., as well as tuberculous glands and other tuberculous foci.

CLINICAL ASPECTS OF INTESTINAL PROTOZOIASIS

By ANDREW BONTHIUS,* M. D., *Pasadena*

Pathogenic parasitic protozoan infections, like bacterial infections, often exist indefinitely in a host without producing symptoms; often they cause such mild symptoms as to escape complaint by the patient; and often the chronic or secondary symptoms are so atypical and indefinite, and so far remote from the actual seat of infection, that the etiologic cause may escape the attention of the physician.

The presence of pathogenic protozoa or their cysts is the only sure proof of the infection.

No patient can be declared cured of intestinal protozoiasis unless, in addition to clinical improvement, the stools remain negative upon successive periodic examinations for at least a year.

DISCUSSION *by H. E. Butka, Los Angeles; R. Manning Clarke, Los Angeles; Herbert Gunn, San Francisco, and John V. Barrow, Los Angeles.*

IN this paper I purpose to discuss chiefly the secondary manifestations, diagnosis, and treatment of intestinal protozoiasis. The parasitic protozoa included in this consideration are the amœba dysenteriæ, balantidium coli, giardia or lamblia, chilomastix, trichomonas, pentatrichomonas, cragia, and councilmania. Each of these, independently or together with one or more of the others, can and often does cause strikingly similar secondary manifestations.

"Entamœbic dysentery is an acute or chronic specific disease of the intestine, caused most commonly by entamœba histolytica, but in some instances, possibly, by other species. These entamœba enter the intestines with food or water and produce colitis and extreme enteritis, characterized by the passage of frequent motions, which generally contain blood and mucus and are associated with abdominal pain and tenesmus. At times they also produce abscesses in the liver and other parts of the body." One may quite correctly add that the above-mentioned parasites, alone or together, may cause the same clinical symptoms.

This definition by Castellani is quite similar to definitions given by several other authors. It is satisfactory for the acute type, but very often the chronic type, or secondary stage, is not characterized by all or even by any of these symptoms. Not every patient whose intestinal tract is invaded by these parasites develops dysentery, severe, mild, or at all. Many go through what may be termed the first stage of invasion by amœba without any, or at most, insignificantly mild, symptoms, and pass on to what I choose to call the secondary stage with its manifestations.

All authorities agree that, under certain conditions, focal infection may exist from one to twenty or more years, during which it may be latent, intermittently active, and later continuously so, depending upon the changed virulence of the invader, and the loss of immunity by the patient, or both. Secondary lesions of focal infection may occur so slowly as to induce no symptoms, and the patient may be unaware of anemia, nephritis, endarteritis, until an advanced stage of invalidism is reached. Rosenow has proved that streptococci in focal infections are not only pathogenic but often possess truly remarkable specificity, for certain organs or cells of the body, as the gall-bladder, stomach, appendix, muscles, kidneys, nerves or heart.

It has been generally accepted as true that bacterial infection may exist indefinitely in a host before producing clinical symptoms and causing symptoms far remote from the actual seat of infection. Why can this not logically hold true for pathogenic parasitic infections also?

From my observations of a series of seventy-four cases with positive parasitic findings, I am of the opinion that bacteria and parasites invade and effect their hosts much alike. This is particularly true of the type of infection most frequently encountered in Caucasians in temperature climates.

My seventy-four patients included those from 3 to 70 years of age; thirty-three were females, forty-one males; forty-nine had lived from one to thirty years in the Orient, two had spent considerable time in Europe, the others had never been out of the United States.

The most frequent complaint offered by these patients was general indisposition, tiredness, exhaustion, lack of energy, in other words — *asthenia.* Fifty-one of the seventy-four gave this as their major complaint. The asthenia is extremely varied in degree and type.

CASE 1—Male; 45; previous history unsuggestive. He complained of "all-tired-out" feeling, a "lead cap" headache and indigestion with distress after eating and loss of appetite.

Physical examination revealed no positive evidence of trouble other than severe pain on deep gentle pressure in the gall-bladder region and a generalized mild icterus. Microscopic examination of feces revealed councilmania.

The patient was given the Weir-Mitchell rest treatment

*Andrew Bonthius (314 Professional Building, Pasadena), M. D. Northwestern University, Chicago. Practice limited to internal medicine. Hospital connections: Los Angeles General Hospital, Pasadena Dispensary. Appointments: Consulting surgeon, U. S. P. H. S., Ancoy, China, 1910-1913; Hope and Whilelmenia Hospitals, Ancoy, China, 1910-1913; Broadway Dispensary, University of California, Los Angeles.

for five weeks under the most favorable conditions and surroundings. At the same time, specific treatment (described later) for parasites was carried out. The patient made some, but not a complete recovery. The lead cap headache almost entirely left and strength returned to a large degree. But icterus and pain in the gall-bladder region continued as before. The gall-bladder was removed and found to be a chronic cholecystitis. No parasites were found in the gall-bladder contents.

This case is of interest because of the lead cap headache, which was very distressing; and because of chronic cholecystitis, which quite probably was secondary to the primary parasitic infection of the intestine. Had I not given the specific treatment before the cholecystectomy was performed, I might have found the parasites in the gall-bladder contents. As it was, the intestinal infection was cleared up and the effects of the gall-bladder infection persisted, just as is usually the case when chronic cholecystitis is secondary to a typhoid infection decades before, or to pneumonia or appendicitis or any focal infection anywhere else.

In this series I have encountered three patients with almost unbearable headache. Other patients developed a secondary cholecystitis, and one appendicitis. In the latter instances the diseased parts had to be resected after the specific treatment was completed. The specific treatment usually will eradicate the parasites, but it cannot correct the pathologic results caused by them.

CASE 2—A physician, 50 years of age, gave a history of having had an attack of dysentery years ago, while residing in the Orient, but had since then been constipated most of the time. His only complaint was a lack of ambition and the almost constant desire to sleep. Past and present history were otherwise negative.

Chilomastix and entameba dysenteriæ were found. Specific treatment was given and the patient states that he is now enjoying his work to the full and the morbid somnolence has left him.

CASE 3—A nurse and the mother of two children. She had lived in the Orient. She was rugged and apparently quite well, but complained that she had not felt equal to her domestic responsibilities for the past year and tired very quickly, which she had never done before. She gave no history of diarrhea.

Giardia, entemeba dysenteriæ, councilmania and trichuris were found. Specific treatment was given and now she feels fully recovered.

CASE 4—Governess, 66 years of age, had traveled extensively for years in this country and in Europe. A physician in another city had told her she probably had a neoplasm in her abdomen and should have an x-ray. Because she preferred to be cared for in Pasadena he referred her to me. I found her past history negative and the physical findings negative with the exception of a decided thickening of the walls of the palpable parts of the colon. She came stating that she felt so "all-in" that she didn't care to live any longer. After specific treatment was started and nearly completed, she was able to resume her usual work with normal vigor.

Osteoarthritis is a symptom commonly encountered.

CASE 5—A Mexican woman, about seventy, with advanced articular deformities of hands and feet, accompanied by almost constant rheumatic pain flashing up and down her arms and legs, obviously of long standing.

Councilmania and entameba dysenteriæ were found. The specific routine treatment has been completed and she reports some relief from the pain. However, it is very questionable that we can give this patient any permanent relief. Her treatment will be continued over a period of time longer than usual and the results noted.

This is the type that Ely classifies as the second great type of chronic arthritis. It is characterized by cartilaginous and bony outgrowths at the margins of the articular cartilages. The disease is common and is known by many names, such as osteoarthritis, hypertrophic-arthritis, degenerative arthritis, senile arthritis. Ely states that "chronic arthritis of the great second type is caused by some form of non-bacterial organism, probably a protozoan, which, domiciled in the gastro-intestinal tract, gains access to the system through the foci of osteomyelitis about the roots of the teeth and causes an aseptic necrosis in the marrow in the region of the joints." The striking feature about this series is not the presence, but the absence of dysenteric symptoms following inflammation of the large or small intestine or both. In fact, only one of my patients had these classical symptoms, which in this instance were caused by chilomastix. A second patient gave a history of alternating periods of loose movements and periods of well-formed ones. A third gave a history of large mucous casts passing frequently, but she was habitually constipated. The remaining seventy-one, with only a few exceptions, stated that they were constipated. The term, dysentery, is a misleading one and a misnomer when applied to the secondary stage of these infections.

The most important finding is that pathogenic varieties of protozoa were present in every patient. But so are some of these parasites present in apparently normal individuals without producing symptoms, you may say. That is true and so are there individuals, enjoying apparent good health, who are the hosts to the spirochetes, diphtheria bacilli, tubercle bacilli, or other microbes. The resistance of an individual to a given infecting agent does not assure the nonpathogenicity of that same germ when invading another host, particularly one with a low resistance.

It is not always easy to find the parasites. If a warm stool can be examined, and the ameba or flagellates can be seen in motion, the diagnosis is easy. This, however, is often impossible, often unreliable and unnecessary. The specimens may be examined cold and the cysts be seen with eosin iodine stain. But this necessitates some precaution. The specimen should be kept in a uniform moderately warm temperature and in a moist condition. The ameba die quickly when exposed to the open air and some cysts disintegrate very quickly under atmospheric and thermal changes and putrefactive gases.

The specimens of my patients are all examined in my own laboratory. The fresh specimens are examined and permanent slides are made for more careful study. Specimens are also sent to Professor Kofoid for a check on my work. I am greatly indebted to Professors Kofoid and Swezy, with whom I studied, for my training and for their unselfish assistance in differential diagnosis of the parasites.

Several times when my findings were positive, negative reports were returned from Kofoid; and there were instances when I did not find any parasites, but Kofoid did. At first, I concluded that I had made a mistake, which is easy to do, because it is not always easy to differentiate an ameba dysenteriæ from a coli, beutschilli or some others.

One patient had a heavy infection of very motile chilomastix. The six consecutive specimens were

heavily loaded. Kofoid reported negative for all of them. Six specimens from each of eight other patients were found positive in my laboratory and negative by Kofoid. Later, I sent a permanently stained slide made from the fresh specimens of each of these eight patients, and one additional one, to Kofoid's laboratory; they reported that five of the nine specimens were positive and the other four slides were not stained satisfactorily.

The discrepancies between the reports from the University of California laboratories and my own are no reflection on the reliability of the work in either instance. It only means that the parasites often die or their cysts disintegrate easily under changing conditions, and, therefore, the sooner the specimen can be examined the more reliable will be the findings.

Then, too, it is important to bear in mind that not every intestine infected with parasites yields parasites or their cysts with every bowel movement. Frequently a patient may present entirely negative specimens for days and then, without any known cause, the specimen will again be heavily infected. One patient whose symptoms and clinical history were quite positively due to parasitic infection, had negative specimens for twenty-two consecutive days. The twenty-third day the specimen was heavily infected with chilomastix. One or two negative specimens are not sufficient for a diognosis of the absence of intestinal parasites.

The treatment of patients with intestinal protozoiasis should be by the intensive employment of some form of ipecac and arsenic in conjunction with thorough bowel irrigations.

Ipecac may be given in powdered form; in capsules, salol coated; in fluid extract—drachm doses—through the duodenal tube; or in the form of emetine hydrochloride—one-third gr. to two-thirds gr. doses—subcutaneously, intramuscularly or intravenously. Intravenously is the best method and the least annoying to the patient. The dose should be given once daily for twelve or fourteen days in succession. It should be borne in mind, however, that ipecac or its derivaties, has no therapeutic value in the treatment of the flagellates.

It is well, at the beginning of the treatment, to inform the patient of the toxic effects of ipecac, because patients commonly complain of a general more or less extreme fatigue, which may develop into a real myalgia, especially of the leg muscles. Frequently patients complain of severe sacro-iliac distress. Two of my patients developed a severe cardiac myalgia and insufficiency. The above mentioned symptoms usually begin to appear about the second week of the ipecac treatment and begin to disappear soon after the administration of the drug is discontinued, though for weeks later the patient may still complain of a degree of this distress. Some patients needed to be confined to their beds during treatment.

After the course of ipecac has been completed, arsphenamine or neoarsphenamine is administered intravenously, beginning with 0.30 gm. and increasing to from 0.60 to 0.90 gm. From four to six doses are given from five to seven days apart.

Some have given neoarsphenamine through the duodenal tube for the treatment of giardia. Mehrtens has shown that when doses, 4.0 gm. are given in the rectum a very large amount is absorbed into the blood and eliminated in the urine. Some treat children by giving the arsphenamine per rectum. Kantor believes that we have in arsphenamine or neoarsphenamine therapy a precise and effective method of reaching susceptible protozoan parasites no matter how deeply they invade the intestinal or biliary mucosa.

Bismuth emetine iodide is given in 3.0 gr. doses nightly until 42.0 grs. are taken. This is best dispensed in double capsules, salol coated, and taken at bedtime. This drug may be given simultaneously as the course of arsphenamine is being given or after it is completed.

The use of mercurochrome in 3.0 gr. doses daily for from ten to fourteen days has been reported as a successful method of treatment.

Enema containing parasiticidal drugs such as quinine, camphor bichloride and coal-oil, have been recommended. If we could be certain that the parasites confined themselves to the large bowel, there might be considerable good results obtained from such enemas. But it is well known that these parasites infect any or every portion of the intestinal tract, including gall-bladder and appendix.

Therefore, I prefer to use duodenal irrigation, not lavage but irrigation, using one and a half quarts physiologic salt solution. This is given every day for three weeks, every other day for three weeks, twice a week for two weeks, and then indefinitely once a week if the patient is troubled with constipation. This method flushes the entire intestinal canal, small and large, in about half an hour and does not exhaust the patient. This is a practical procedure from the standpoint of the patient. Every patient, almost without exception, can be taught to swallow and pass the tube into the duodenum, and then, to take the duodenal irrigation at home.

After the treatment has been completed examinations of the stools should be repeated. If a series of at least six specimens be negative, further examination may be postponed for three months. If, by then, the parasites and symptoms have reappeared, the routine treatment should be repeated. If the symptoms have completely disappeared or have been arrested and series of examinations at three months' intervals over a period of a year are negative, the patient may be declared cured.

It is not uncommon to find the parasites again several months after a course of treatment has been completed. Frequently it will be necessary to keep the patient under treatment for from six to twelve months. The treatment of patients infected with intestinal protozoa often must be intensive, persistent and extended, in order to accomplish eradication. Some patients are never completely freed of the parasites; but in spite of this the treatment may effect marked improvement.

1. The secondary manifestations of protozoan infection vary widely.

CONCLUSIONS

2. The presence of the pathogenic parasites or their cysts is the only sure proof of the infection. A series of not less than six specimens should be

examined before a negative diagnosis is considered established.

3. The recognized specific treatment is ipecac and arsenic in their various forms, and intestinal flushing over a definite period of time.

4. No patient can be declared cured unless the symptoms have discontinued and the stools remained negative over a period of a year.

5. One course of treatment may not effect complete eradication of the parasite.

6. Protozoan infections must be considered in our differential diagnosis just as bacterial infections are.

DISCUSSION

H. E. BUTKA, M. D. (White Memorial Hospital, Los Angeles)—The subject of intestinal parasites especially appeals to me. Opinions as to the role of the flagellates and certain ameba varies. It is not difficult to convince ourselves of the pathogenicity of the various cestodes and nematodes and flukes when found in the intestinal tract, yet we find many times these parasites may not produce clinical symptoms. But when we come to the smaller microscopic parasites the same difficulty exists as once existed in convincing the world of the pathogenicity of certain bacteria.

My experience with intestinal parasites during the last few years convinces me that in most instances there are definite clinical symptoms associated with the presence of these parasites. These may be manifested by diarrheal conditions, gastro-intestinal disorders of a periodic type, neurologic symptoms, or general constitutional symptoms. As in infestation with entameba histolytica, there are frequently periods of inactivity on the part of the parasites, and it is these periods of absence of symptomatology that some physicians point to as proof of their non-pathogenicity.

R. MANNING CLARKE, M. D. (606 South Hill Street, Los Angeles)—I have very much enjoyed this excellent paper by Doctor Bonthius, which so ably discusses the intestinal protozoa. The question of diagnosis is always a hard one in these cases, and I agree with Bonthius, in the matter of urging frequent and repeated examination of stools. It often happens that one examination of a stool is made and a diagnosis of no parasites given. This is certainly preposterous. I have enjoyed this evening hearing Bonthius urge the matter of repeated and frequent stool examinations before it is decided that there are no parasites present.

Regarding the pathogenicity of some ameba there is no difference of opinion. However, regarding some of the flagellates, such as the chilomastic, there is some difference of opinion regarding their pathogenicity. I know of many most able writers who believe that such parasites as the chilomastix are present as some result of some other condition that excites inflammation or trouble in the tract. For instance, I have seen patients with ulcer put on Sippy management, keeping the hydrochloric acid controlled by alkaline powders given by mouth. After this has attained for a certain length of time, I have seen chilomastix and other flagellates appear in the stools only to disappear again after the alkaline treatment had been stopped and the hydrochloric acid allowed to reappear in the stomach.

I recently had a case of amebic dysentery that had also a large number of chilomastix. As soon as the ameba disappeared there was no more trouble with the chilomastix. In British literature this idea prevails more than it does among American writers. Personally, I am rather inclined to their point of view, although I am not prepared to back it up entirely. There is much more yet for all of us to learn regarding this subject.

I feel there is nothing to be added to what Bonthius has given us about treatment. Certainly stools should be examined repeatedly and painstakingly. To give a patient one round of emetine treatment and turn him loose with the assurance that he is well is wrong. All of us have seen this done and have also encountered the resulting disappointment on the part of the patient.

HERBERT GUNN, M. D. (350 Post Street, San Francisco)—During the past few years considerable interest has been evinced by the medical profession in the subject of protozoan infections of the intestinal tract and much has been added to our knowledge of the subject. However, nothing has been adduced up to the present time to warrant the assumption that all protozoa found in the stools are pathogenic. The pathogenicity of entamœba histolytica is, of course, well recognized.

Entamœba coli is generally accepted as being non-pathogenic, which is, I believe, usually the case. Occasionally entamœba coli infections apparently produce symptoms usually of a non-dysenteric character which are relieved by eradication of the parasites. However, in such cases the possibility of the presence of an undemonstrated entamœba histolytica must be taken into consideration.

The ameba referred to by Bonthius as a councilmania can safely be considered as a coli. The flagellates are, in my opinion, usually harmless and if any symptoms are produced by them they are transitory and indefinite. There certainly is no warrant for the wholesale incrimination of these parasites for the numerous symptoms often ascribed to them. It is fortunate that the flagellates are practically harmless for we have had no drugs which will destroy them.

There is certainly not the slightest foundation for the belief that arsphenamine or neoarsphenamine combined with ipecac, or its derivatives, has any destructive effect on intestinal protozoa other than amebæ.

During the past twelve years I have used ipecac and emetin combined with salvarsan and neosalvarsan for the treatment of amebiasis on several hundred cases, and these drugs have not had the slightest effect on any of the flagellates as far as their eradication was concerned.

It may be possible that treatment is indicated in individual cases for flagellate infections, but I can find no justification for the general treatment of all such cases.

JOHN V. BARROW, M. D. (2007 Wilshire Boulevard, Los Angeles)—I wish to welcome Doctor Bonthius' paper on protozoa and to emphasize a few of the points we have been struggling to bring out for years.

There is not much criticism to be offered in his presentation. The subject matter brings up points of disagreement among clinicians because of the varying clinical experiences each one has had in the treatment of these diseased persons. The clinical picture drawn by Castellani can certainly be produced by several of the protozoa, either singly or collectively, and the clearing up of these infections results in better health to everyone treated.

I am sorry Bonthius has not given the blood pictures in his cases. The white cell count with the differential, in my experience, is certainly significant as has been recorded in the literature previously.

There is no doubt that arthritic cases improve generally in proportion to the success we have in ridding them of these parasites. I think the argument here is logical and based on correct clinical facts. We must wait for chemistry and biology to prove this point. The only facts we have at present are clinical. There is not one vestige of laboratory work against the contention. Clinical evidence should have the right-of-way until there is at least some other evidence against it. Like Mark Twain's death, the myocardial weakness produced by ipecac and emetin has been very much exaggerated. I certainly do urge the treatment of all protozoan cases, flagellates, as well as the amœbas. The fact that the organisms are difficult to eradicate is taken to mean that they are non-pathogenic, but it must be remembered that certain spirochetes and tubercle bacilli are difficult to eradicate.

The symptomatology of protozoan infection is indefinite, but not transitory. The organisms are chronic and the change in the system produced by them is a decided clinical imbalance which clears up generally when the parasites are gone. It is logical that these organisms may be present for years before this imbalance is produced.

I am glad that Bonthius has discussed much of the treatment. I believe the arsenic preparations are destruc-

tive to the giardia in the duodenum. From my experience, neo-arsphenamine into the vein is not particularly harmful to the amœba. Stovarsol internally has been much more efficacious in my cases.

We shall advance rapidly in treatment as well as in pathological proof of the harmfulness of these organisms, when someone cultivates them in pure culture. What we need is their isolation and culture in the same manner we cultivate typhoid and diphtheria organisms. At present the clinical expression of these parasites should lead us in regard to their treatment.

I have no sympathy with the contention that only one organism, the ameba, out of the great group of protozoa can be harmful to mankind. That contention would be analogous to claiming that there is only one insect injurious to farm crops.

RICKETS

Langley Porter, who has been in Europe traveling and studying for over a year, sends the following resumé of our knowledge of rickets.—EDITOR.

Rickets is a disease affecting all the bodily tissues, especially the osseous, the nervous, the muscular, and the epithelial. The changes in the bones are the most visible, especially in the earlier stages of the disorder. For this reason and also because the x-ray can be used to record even the earliest of such changes rachitic anomalies of ossification and of bone structure have been much studied also. The fact that extreme degrees of bony change lead to deformities that interfere with skeletal function lends further interest to studies of the osseous changes in this disease.

The changes in the nervous tissues tend to establish conditions of heightened irritability and diminished inhibition; conditions that are the basis of the clinical entity which is called tetany and whose symptoms are ready access of convulsive seizures, facial irritability, carpopedal spasm, laryngo spasm, emotional instability and anomalies of behavior. These discernable tissue changes are, however, the results of an alteration of metabolism which is revealed by a change in the ratio of calcium to phosphorus in the blood serum and by a decrease in its contained bases, a relative acidosis. This metabolic imbalance is brought about by some noxious influence acting on the rapidly growing tissues. Present opinion holds that this noxious influence is permitted to act because the body receives an insufficient stimulation of radiant energy, especially of that form of radiant energy which emanates from the violet part of the spectrum in waves of a length close to 300 mm. Such radiant energy appears to be essential in order that the chemistry of the tissues should proceed with optimum energy and the metabolism produce normal growth.

The radiant energy is received directly from the impingement of sunlight on the body and indirectly from accessory foodstuffs, especially the fats and the green leaf vegetables in which it is stored in static form. After digestion, metabolism has the power to transform this static energy to dynamic in a form that acts on the chemical events that happen in the body in such a way that it increases their speed and vigor. Apparently, through the aid of these accessory foodstuffs which we call vitamines, the whole series of oxidations and reductions which constitute life is speeded up and perfected. Apparently each grown individual has his own quantitative need for such radiant energy. If he fails to receive all that he needs, he develops rickets.

In most latitudes under usual conditions of housing, climate and clothing, the direct radiations that reach the growing human do not suffice to supply his optimum needs, therefore, it becomes necessary to make up the lack by the ingestion of such foodstuffs as have the radiant energy stored in the forms we call vitamines. An especially rich source lies in cod-liver oil. The fats of milk, from properly housed and pasture-fed cattle, and the green leaf vegetables, are the most dependable and available sources of supply.

Recently, apparently successful attempts have been made to increase the radiant energy content (vitamin value) of such foodstuffs by subjecting them to the influence of violet-rays originating in mercury vapor lamps and quartz lenses. The winter season and substances suspended in the atmosphere such as fog, cloud, smoke and rain, may prevent a large part of the violet emanations from reaching the human body. Window-glass and the usual clothing provide further hindrances. For these reasons it is necessary, especially during the winter months, to insure to the growing child a full supply of vitamine containing foods. This we can do most certainly by feeding cod-liver oil in addition to milk fat, green leaf vegetables, egg yolk, and the juice of the citrus fruits, and the tomato. Heliotherapy, or radiation with mercury vapor lamps, will give an added insurance against the development of rickets. It must not be forgotten, however, that the ingestion of an ample supply of antirachitic vitamine is no certain preventive of rickets. For the patient may be subject to disturbances of digestion, of assimilation that interfere with the proper absorption and the utilization of vitamines. On the other hand, he may be the victim of some parental infection or intoxication which hinders that acceleration or stepping up of metabolism which is normally consequent upon the influence of radiant energy, whether it be derived directly from light or indirectly from the supplies stored in the vitamine carrying foods. The consideration and cure of all such illness is imperative in all attempts to prevent or to cure rickets.

384 Post Street.

The Doctors and Other Citizens of Oakland and Alameda County are hosts to the California Medical Association at this year's session which will be held April 26 to May 1, 1926, inclusive. The interesting program which is being prepared provides for a whole week of social pleasures and scientific work. This ought to be a banner meeting of the State Association.